Biochem Biophys Res Commun. 2018 Jan 22;495(4):2462-2468. doi: 10.1016/j.bbrc.2017.12.146. Epub 2017 Dec 27.

Hydrogen postconditioning promotes survival of rat retinal ganglion cells against ischemia/reperfusion injury through the PI3K/Akt pathway

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PMID: 29288664 DOI: 10.1016/j.bbrc.2017.12.146

Abstract

Retinal ischemia/reperfusion injury (IRI) plays a crucial role in the pathophysiology of various ocular diseases. Our previous study have shown that postconditioning with inhaled hydrogen (H_2) (HPC) can protect retinal ganglion cells (RGCs) in a rat model of retinal IRI. Our further study aims to investigate potential mechanisms underlying HPC-induced protection. Retinal IRI was performed on the right eyes of rats and was followed by inhalation of 67% H_2 mixed with 33% oxygen immediately after ischemia for 1 h daily for one week. RGC density was counted using haematoxylin and eosin (HE) staining, retrograde labelling with cholera toxin beta (CTB) and TUNEL staining, respectively. Visual function was assessed using flash visual evoked potentials (FVEP) and pupillary light reflex (PLR). The phosphorylated Akt was analysed by RT-PCR and western blot. The results showed that administration of HPC significantly inhibited the apoptosis of RGCs and protected the visual function. Simultaneously, HPC treatment markedly increased the phosphorylations of Akt. Blockade of PI3K activity by inhibitors (LY294002) dramatically abolished its anti-apoptotic effect and lowered both visual function and Akt phosphorylation levels. Taken together, our results demonstrate that HPC appears to confer neuroprotection against retinal IRI via the PI3K/Akt pathway.

Keywords: Apoptosis; Hydrogen; Ischemia/reperfusion; PI3K/Akt pathway; Postconditioning.

Related information

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Gene (GeneRIF)
Nucleotide (RefSeq)
Nucleotide (RefSeq)
Nucleotide (Weighted)
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